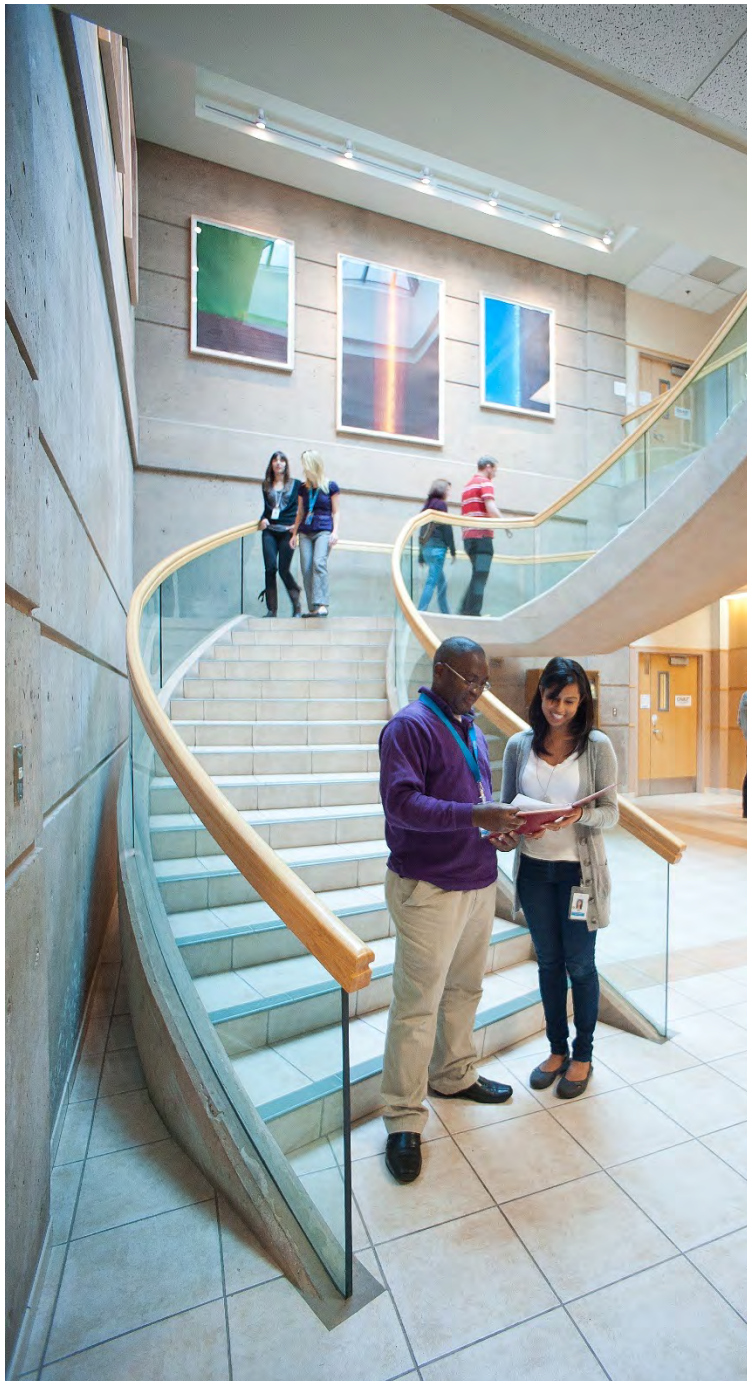
2023 Summer Student Poster Day



Attendees are encouraged to ask questions!

To ask a question use the chat feature or unmute your mic during the designated Q&A period

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Session #1

Maisa Samiee
Natasha Kaprelova
Ariel Qi
Emily Simpson
Venessa Thorsen
Rory Trevorrow
Amrith Vincent
Thumri Waliwitiya

#2

Maisa Samiee

Undergraduate Student, University of British Columbia

Supervisor: Simon Massey

Monitoring Antagonism of Neuromuscular Blockade: BCWH Current Practice Compared to the American Society of Anesthesiologists 2023 Practice Guideline: a retrospective clinical audit

Abstract & Poster - https://bcchr.ca/posterday

#3

Natasha Kaprelova

Master's Student, University of British Columbia

Supervisor: Chinten James Lim

Investigating MYC Amplification in IL-6/JAK/STAT3-Mediated Treatment Resistance in Group 3 Medulloblastoma

Abstract & Poster - https://bcchr.ca/posterday

Investigating c-Myc Amplification in IL-6/JAK/STAT3-Mediated Chemoresistance in Group 3 Medulloblastoma

Natasha Kaprelova[1,3], Chinten James Lim[2,3]

[1]Dept of Medicine, [2]Dept of Pediatrics, University of British Columbia

[3]Michael Cuccione Childhood Cancer Research Program, BC Children's Hospital Research Institute, Vancouver, B.C., Canada

Background and Introduction

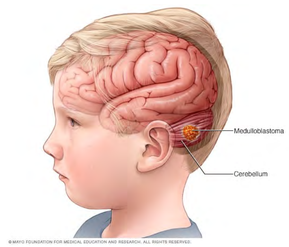


Figure 1: Medulloblastoma is believed to originate from progenitor cells in the cerebellum

- Medulloblastoma (MB) is the most common malignant brain tumour diagnosed in children, accounting for 20% of all childhood brain tumours.
- MB was once believed to be a single disease entity, but transcriptional profiling studies have revealed existence of 4 subgroups: WNT, SHH, Group 3 and Group 4. Each subgroup exhibits distinct biology, prognosis and risk implications.
- Group 3 MB is the most aggressive subgroup and has the worst prognosis. The lack of effective therapeutic treatments for this subgroup due to a limited understanding of its molecular pathways necessitates further studies to understand the mechanisms of tumor progression and aggressive tumor behavior.

MB Subgroup	WNT	SHH	Group 3	Group 4
% of cases	~10%	~30%	~25%	~35%
Age at diagnosis				
Gender	♂:♀	♂:♀	♂♂:♀	♂♂♂:♀
Prognosis				
Histology	Classic	Desmoplastic, Classic, LCA	Classic, LCA	Classic, LCA
Metastasis	5-10% Rare, Local	15-20% Local	40-45% Distant	35-40% Distant
Cell origin	Progenitor cells in lower rhombic lip	Granule precursors of external granule layer	Neural stem cells	Unipolar brush cells
Recurrent gene amplification	-	MYCN, GLI1 or GLI2	MYC, MYCN, OTX2	SNCAIP, MYCN, OTX2, CDK6
Recurrent SNVs	CTNNB1, DDX3X, SMARCA4, TP53	PTCH1, TERT, SUFU,SMO, TP53	SMARCA4, KBTBD4, CTDNEP1, KMT2D	KDM6A, ZMYM3, KTM2C, KBTBD4
Cytogenetic events (Gain / Loss)	6	9q,10q,17p / 3q, 9p	8,10q,11,18q / 1q,7,18 / i17q	8, 11p, X / 7,18q / i17q
Infant Child Adult Male Female Prognosis indicator (Excellent Intermediate Poor)				

Figure 2: Molecular subtypes of Medulloblastoma

Hypothesis/Aims/Experimental Approach

The IL-6/JAK/STAT3 signaling pathway that mediates the acquired chemoresistance in the most aggressive subtype of MB (Group 3) is dependent on c-Myc amplification and function

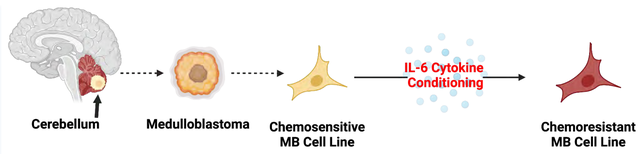
Aim 1: Establish the Requirement of MYC in IL-6/STAT3 Mediated Chemoresistance

1. Compare c-Myc levels prior to and after 4 weeks of conditioning with IL-6 in Group 3 MB cells
2. Knockout MYC using CRISPR to derive a Med8A MYC$^{-/-}$ cell line and assess if loss of MYC abrogates IL-6 mediated drug resistance
3. Use MYC inhibitors (10058-F4 & 10074-G5) to disrupt MYC/MAX dimerization and impair MYC gene expression

Aim 2: Assess if Combination of MYC Inhibitors with Vincristine will Overcome Chemoresistance

1. Establish the maximal tolerable dose of inhibitors without inducing cell death
2. Assess if prophylactic treatment of appropriate dosage throughout the 4-week IL-6 conditioning period can suppress development of drug resistance
3. Assess if combined treatment with subtoxic levels of MYCi with vincristine for 48 hrs can be effective in overcoming pre-established drug resistance in Grp3 MB cells

Modeling Chemoresistance



Preliminary Findings

IL-6/JAK/STAT3 Signaling Pathway Drives Chemoresistance in Group 3 Medulloblastoma

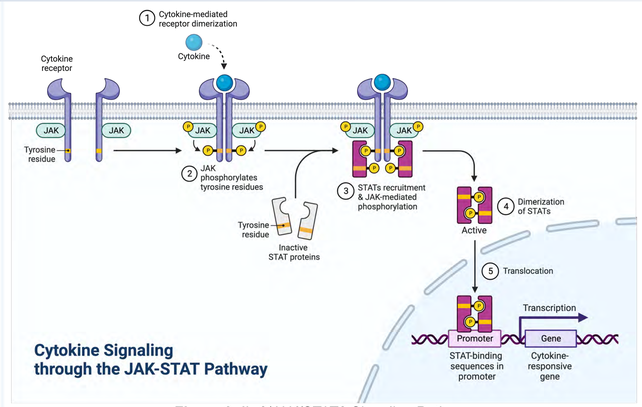


Figure 3: IL-6/JAK/STAT3 Signaling Pathway

Exogenous IL-6 conditioning promotes drug resistance in Group 3 MB cell lines

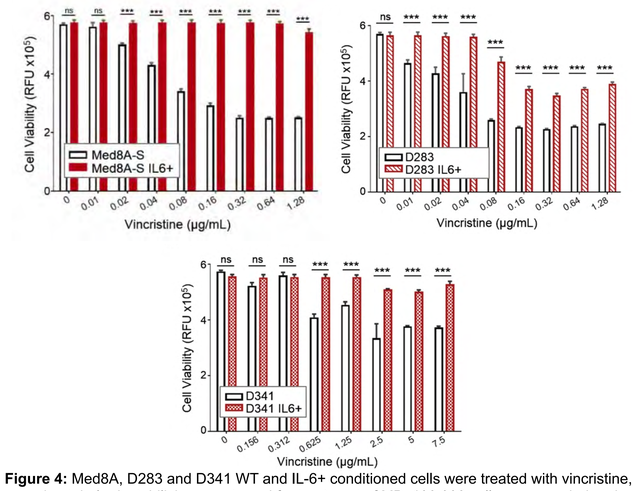


Figure 4: Med8A, D283 and D341 WT and IL-6+ conditioned cells were treated with vincristine, a microtubule destabilizing agent used for treatment of MB. 100,000 cells were seeded and incubated with indicated concentrations of drug for 48 hours. Cell viability was assessed by measuring the fluorescence after incubation with CTB for 4 hours.

Exogenous IL-6 conditioning results in increased levels of c-Myc

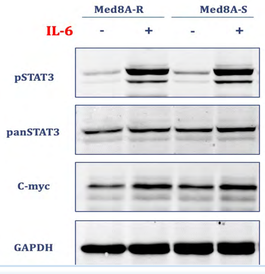


Figure 5: Med8A-S (chemosensitive) and Med8A-R (chemoresistant; derived via selective conditioning with vincristine) were examined for the expression of total STAT3 (panSTAT3), phospho-STAT3 at residue Y705 (pSTAT3) and total c-Myc by Western Blot analysis under basal conditions and following IL-6 conditioning.

Anticipated Results

- Preliminary data suggests that amplification of c-Myc in Group 3 Medulloblastoma is dependent on IL-6 cytokine levels being secreted in the tumour microenvironment.
- I anticipate that following knockout of IL-6/JAK/STAT3 signaling components (IL-6R, gp130 and STAT3), cells conditioned with IL-6 cytokine will still reveal upregulated expression of c-Myc levels relative to basal conditions. This discovery can reveal a significant finding in that IL-6 cytokine can signal through a non-IL6R, proposing a novel signaling mechanism in the IL-6/JAK/STAT3 signaling pathway.
- I anticipate that CRISPR-mediated knockout of c-Myc will decrease Group 3 MB cell lines' chemoresistance to vincristine, highlighting c-Myc as a crucial component in driving drug resistance.

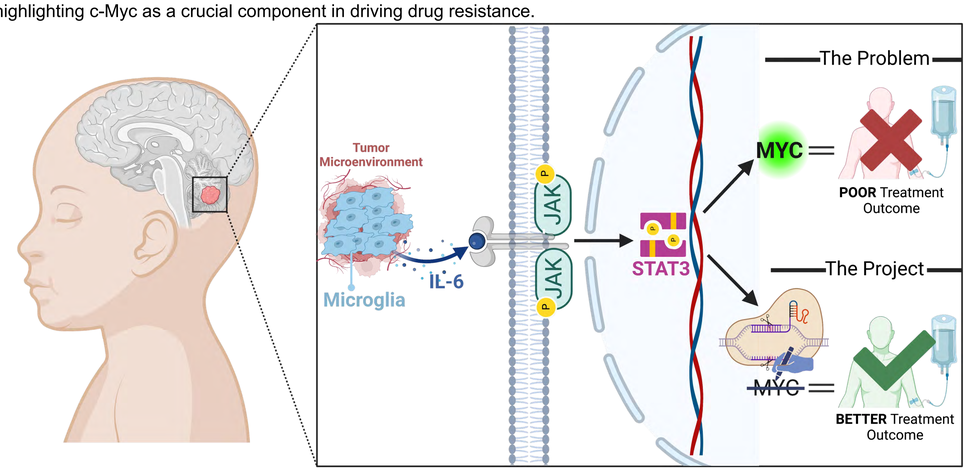


Figure 6: Simplified overview of research proposal

Current Challenges/Conclusion

- MYC is a proto-oncogenic transcription factor that belongs to the basic-helix-loop-helix-leucine zipper (bHLHZip) family and encodes a nuclear phosphoprotein that is involved in the transcription of genes that regulate the cell cycle, proliferation, embryonic development, and apoptosis.
- Expression of MYC factors is tightly controlled throughout the cell cycle, but may become dysregulated, leading to malignant transformation.
- c-Myc is an Intrinsically Disordered Protein (IDP), and therefore its extended unstructured surface lacks the required "hotspots" and deep hydrophobic pockets that are typically targeted effectively using conventional small molecule drugs.
- To date, no small molecule inhibitors that directly target the c-Myc/Max interaction have progressed to clinical trials.
- Our current proposal examines the IL-6/JAK/STAT3 signaling pathway as the main pathway leading to amplification of c-Myc and sequentially the development of drug resistance of Group 3 MB tumours.
- Using CRISPR-Cas9 gene editing and commercially available inhibitors to target c-Myc, we will elucidate the role of c-Myc in the most aggressive subgroup of MB.

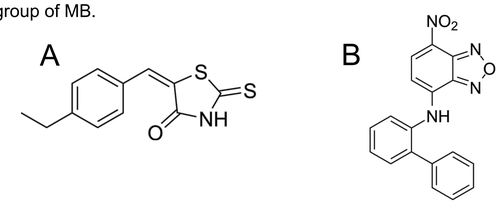


Figure 7: Using literature, we selected 2 conventional small molecules that have been identified to inhibit c-Myc/Max dimerization or DNA binding 10058-F4 (left) and 10074-G5 (right)

Significance and Future Directions

The current proposal addresses the molecular mechanisms of IL-6/STAT3 signaling uniquely implicated in acquired drug resistance in c-Myc-amplified Group 3 MB cells. The expected research findings will identify molecular therapeutic targets that can be used to inform development of novel strategies to combat pediatric brain tumors with the worst prognosis and offer novel pre-clinical insight into efficacy for targeting c-Myc, paving the way for the development of the first effective treatment that targets c-Myc

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#4

Ariel Qi

Medical Student, Queen's University

Supervisor: S. Evelyn Stewart

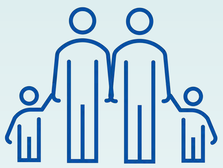
Exploring Family Factors in Pediatric Obsessive-Compulsive Disorder and Psychiatric Outpatient Controls

Exploring Family Factors in Pediatric Obsessive-Compulsive Disorder and Psychiatric Outpatient Controls

Ariel (Ruo Chen) Qi [1,2], John R. Best [2,3], Gordon Andjelic [2,3], Anna MacLellan [2,3], Boyee Lin [2,3], Cynthia Lu [2,3], S. Evelyn Stewart [2,3,4]

[1]Faculty of Medicine, Queen's University; [2]British Columbia Children's Hospital Research Institute; [3]Department of Psychiatry, University of British Columbia; [4]British Columbia Mental Health and Substance Use Research Institute

Background



Youth psychiatric illness can place a heavy burden on the family. In turn, the family environment could impact prognosis and treatment adherence in the youth. Thus, consideration of **family factors** is critical in the context of pediatric psychiatric illness.

Family functioning impairment has been well characterized in pediatric obsessive-compulsive disorder (OCD), with profound impacts on family routines, socio-occupational factors and emotional responses.[1]

In pediatric OCD, poor **parental tolerance of child's distress (PTCD)** and accommodation behaviors are associated with increased symptom severity and treatment resistance.[2]

Proposed Study

Rationale:

There remains a need to characterize how family functioning and PTCD in OCD compare to that experienced in families coping with other youth psychiatric conditions.

Objectives:

1. Compare various aspects of family functioning impairment and PTCD between pediatric OCD and non-diagnostically selected psychiatric controls

2. Explore how patient/family characteristics influence the degree of disease-related family functioning impairment and PTCD

Methodology

Data:

- **Family Input Tool** at BC Children's Hospital (n=4009, years 2019-2023)
  - 6 outpatient psychiatry clinics
- **OCD registry** of the Provincial OCD Program (n=398, years 2011-2020)

Predictors of Interest:

- Patient demographics, psychiatric illness (OCD vs. non-OCD), and medical history
- Patient's family environment
- Family's medical history (psychiatric diagnoses in particular)

Outcome Measures:

- 21-item **Family Functioning Impairment Scale** (modified from the validated OCD Family Functioning Scale)[1]
- 3-item **PTCD Scale** (modified from the validated Distress Tolerance Scale)[3]

Statistical analysis:

- Multivariable linear regression models, whereby the family-related outcome score is regressed on clinical predictors and covariates

This project has been approved by the UBC Research Ethics Board.

Results

Characteristic	**Non-OCD**, N = 3,748[1]	**OCD**, N = 652[1]
Sample		
Family Input Tool	3,748 (100%)	254 (39%)
OCD registry	0 (0%)	398 (61%)
Age	12.5 (3.5)	13.8 (3.1)
(Missing)	1,416	147
Gender		
Female	967 (41%)	246 (55%)
Male	1,251 (54%)	196 (44%)
Other	119 (5.1%)	8 (1.8%)
(Missing)	1,411	202
Ethnicity		
Non-White	389 (18%)	69 (14%)
White	1,700 (79%)	397 (82%)
Other/Don't know	76 (3.5%)	19 (3.9%)
(Missing)	1,583	167
Highest Parental Education		
Less than university degree	783 (36%)	150 (30%)
University degree or greater	1,352 (62%)	337 (68%)
Other/Not Sure	44 (2.0%)	5 (1.0%)
(Missing)	1,569	160
Parental Marital Status		
Married/common-law	1,536 (70%)	393 (79%)
Separated/Divorced	434 (20%)	82 (16%)
Other/Not Sure	210 (9.6%)	22 (4.4%)
(Missing)	1,568	155
[1] n (%); Mean (SD)		

Table 1. Participant characteristics breakdown based on sample source, age, gender, ethnicity, parental education status and marital status

Significance

This project will characterize and compare family functioning impairment and PTCD in non-OCD youth psychiatric disorders in relation to OCD.

Results will help identify populations of patients who **experience greater family dysfunction.**

Our findings could also **inform targeted interventions, treatment strategies and social supports** that are catered to the unique circumstances of the youth patients and their families.

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Acknowledgements

BC Children's Hospital™ Provincial Health Services Authority

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#5

Emily Simpson

Medical Student, University of British Columbia

Supervisor: Christine Voss

Sleep quality in children and youth with type 1 diabetes: a validation study utilizing commercial activity trackers

Sleep Quantity and Quality in Children and Youth with Type 1 Diabetes: A Validation Study Utilizing Commercial Activity Trackers

Emily Simpson[1,2], Ty Sideroff[1,3], Nick Wall[1], Elizabeth Keys[4], Quevie Reinz Abalde[4], Calli Davidson[1,3], Simran Gill[1,5], Holly Buhler[6], Trent Smith[6], Deanne Taylor[6], Christine Voss[1,7]

[1]Centre for Chronic Disease Prevention and Management, UBC; [2]Southern Medical Program, UBC; [3]School of Health and Exercise Science, UBCO; [4]School of Nursing, UBCO; [5]Women+ and Children's Health Sciences, UBC; [6]Interior Health; [7]Department of Pediatrics, UBC

BACKGROUND

- Over 2,500 children and youth in BC live with diabetes[1]
- Children with type 1 diabetes (T1D) are particularly vulnerable to experiencing disturbed sleep and inadequate sleep has been linked to reduced executive functioning and quality of life[2]

OBJECTIVE

- To establish validity of a consumer wearable, the Fitbit Charge 5, for the assessment of sleep behaviours in children and youth with T1D

METHODS

- An observational longitudinal study assessing sleep via commercial activity trackers and sleep logs over a 7-day period

- **Surveys**: Parent/Guardians completed Demographic Questionnaire and Sleep Disturbance Scale for Children (SDSC)[3]

- **Fitbit**: REDCap extracted min-by-min Fitbit sleep data via a custom written API

- **Sleep Logs**: Participants received an SMS message each morning with a survey link to capture information on bed/wake time, sleep quality, and diabetes indicators

- ***Opt-in***: Some provided access to sleep-relevant CGM data to analyze relationships between sleep data and clinical outcomes

- Agreement in Fitbit and sleep log data was assessed via intra-class correlation coefficients and Bland Altman Analyses

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RESULTS

Table 1. Sample Characteristics

n	8
Age (yrs ± SD)	10.74 ± 3.19
% Girls	38
Time Since Diagnosis (yrs ± SD)	5.87 ± 4.44
A1C (% ± SD)	6.96 ± 0.73
% CGM	88
% Insulin Pump	75
SDSC Total Score (mean ± SD)	48.63 ± 8.63

CGM – Continuous Glucose Monitor; SDSC – Sleep Disturbance Scale for Children

** Data are n, mean, or % ± standard deviation*

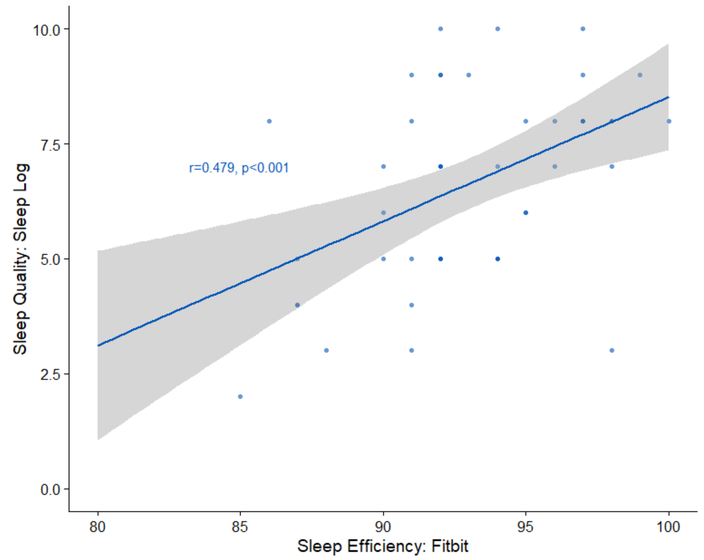

Figure 1: Scatterplot of sleep quality, on a scale of 1 to 10, indicated in sleep logs and sleep efficiency, as a percent, measured by a Fitbit Charge 5

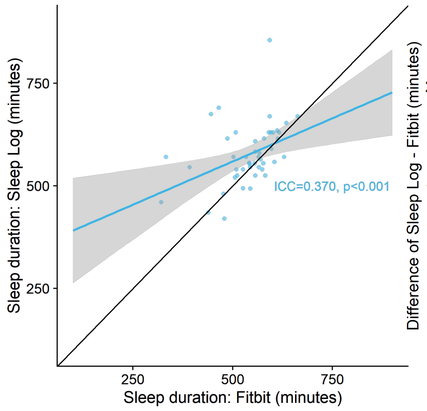

Figure 2: Scatterplot of sleep duration recorded in a log and measured with a Fitbit

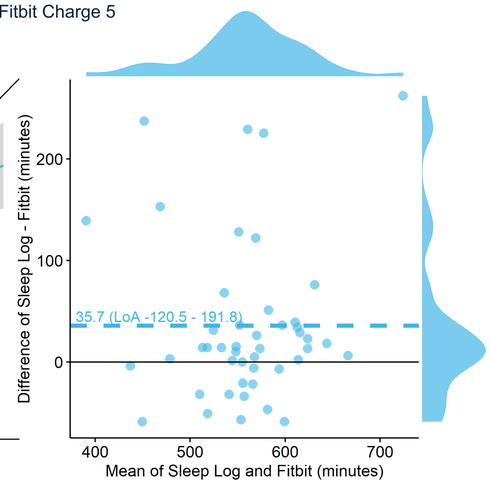

Figure 3: Bland-Altman analysis plot for sleep duration: logs vs Fitbit

Table 2. Sleep metrics and agreement between Fitbit and sleep log

	Persons (n)	Person-Days (n)	Duration/Time of Day (hh:mm)		ICC	Bias* (LoA) in Minutes
			LOG	FITBIT		
Duration	8	46	9:36 ± 1:14	9:01 ± 1:12	0.37**	36 (-120 to 192)
Wake Time			7:44 AM ± 1:08	7:22 AM ± 1:13	0.60***	23 (-97 to 142)
Bed Time			10:06 PM ± 1:21	10:14 PM ± 1:19	0.72***	-8 (-126 to 110)

** Bias indicated as log - Fitbit*

CONCLUSIONS

- There was a moderately strong positive association between self-reported sleep quality and Fitbit-derived sleep efficiency score
- There was significant but poor (duration) to moderate (bed and wake time) agreement between the Fitbit and sleep logs
- Based on preliminary analysis, I believe there is a use for the Fitbit Charge 5 in sleep research for children and youth with T1D

ACKNOWLEDGEMENTS

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Faculty of Medicine

#6

Venessa Thorsen

Master's Student, University of British Columbia

Supervisor: Kevin Harris

Knowledge to Action: Developing a Knowledge Translation Strategy to Improve Management of Familial Hypercholesterolemia in British Columbia

Abstract & Poster - https://bcchr.ca/posterday

Developing a Knowledge Translation Strategy to Improve Management of Familial Hypercholesterolemia in British Columbia

Venessa Thorsen[1], Kevin Harris[1], Stephanie Glegg[2], Jason Sutherland[3]

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[2]Department of Occupational Science & Occupational Therapy, University of British Columbia

[3]School of Population and Public Health, University of British Columbia

BACKGROUND

Children with untreated cholesterol disorders such as Familial Hypercholesterolemia (FH) have an increased risk of heart attack and stroke in early adulthood[1,2]

Lack of clinical practice guidelines for pediatric FH were cited by physicians as a major barrier to management[2]

Updated clinical practice guidelines (2022)[1]

Canadian Cardiovascular Society

Evidence suggests that more than 90% of FH cases are still going undiagnosed[1,2]

Solution: Knowledge translation to physicians

Increased screening and management of pediatric FH

OBJECTIVE/AIM

To increase screening and management of pediatric dyslipidemias amongst family physicians and pediatricians in British Columbia.

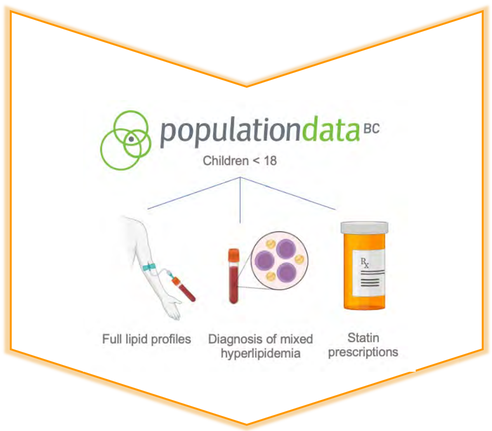
METHODS

Knowledge Translation Intervention

- Case-based learning
- Interactive sessions with polling activities and discussions
- Synchronous/asynchronous delivery
- Based on 2022 CCS guidelines
- Led by pediatric cardiologists
- CPD accredited

One-month Follow-up

- Assess learning, familiarity and confidence managing pediatric dyslipidemias based on responses to patient vignettes
- Self-reported behaviour change
- Satisfaction with the intervention and feedback



Population Data BC pre- and 1 year post-webinar for children <18

Screening: Full lipid profiles
Diagnosis: ICD codes for dyslipidemias
Treatment: statin prescriptions, referral to cardiologist

- Stratified by age, sex, practitioner and patient local health area, specialty of referring practitioner

INCLUSION CRITERIA

Participants who:

- Are licensed family physicians or pediatricians in British Columbia (including academic-based, community-based and subspecialty)
- Routinely care for patients between the ages of 9-11
- Consider themselves to be the primary caregiver for some and/or all of their patients

EXPECTED OUTCOMES

Anticipated sample size of 50-100+ doctors across BC

Increased confidence, learning and self-reported behaviour change after 1 month

Increases in screening, diagnosis and treatment of dyslipidemias across BC at 1 year post-webinar

REFERENCES

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This study is funded by CIHR, the Cordula and Gunter Paetzold Fellowship, and an Evidence to Innovation (E2i) Theme Seed Grant.

#7

Rory Trevorrow

Medical Student, University of British Columbia

Supervisor: Lise Leveille

Perioperative Management of Juvenile Idiopathic Arthritis (JIA) in Anterior Cruciate Ligament (ACL) Reconstruction

UBC Faculty of Medicine, Department of Orthopaedics

Perioperative Management of Juvenile Idiopathic Arthritis (JIA) in Anterior Cruciate Ligament (ACL) Reconstruction

Rory Trevorrow, Daniella D'Amici, Joyce He, Helen Crofts, Hayley Spurr, Kristin Houghton, Lise Leveille

Background

Juvenile idiopathic arthritis (JIA) refers to a group of conditions of unknown etiology characterized by joint inflammation presenting prior to 16 years of age and persisting for a minimum of 6 weeks duration. JIA may co-occur with other musculoskeletal injuries and diseases, including anterior cruciate ligament (ACL) rupture. In the paediatric population, early operative management of ACL rupture is associated with a decreased likelihood of joint instability, pathological laxity, and symptomatic meniscal tears compared to non-operative management. In children with JIA, the inherent inflammatory environment of the joint may interfere with post-surgical healing and rehabilitation. However, medication used to manage JIA, such as corticosteroids, NSAIDs, biological DMARDs, and synthetic DMARDs may also increase complication risk. As such, further understanding of the optimal management of disease activity in the perioperative period may be critical to improve surgical outcomes and reduce complication rates following paediatric ACL reconstruction.

Objective

The aim of the present study is to determine best practices for the medical management of JIA during the perioperative period of paediatric ACL reconstruction.

Methods

Existing literature on the management of inflammatory arthritis in the perioperative period was gathered through a structured search of MEDLINE, Embase and CINAHL databases. A combination of keywords and subject headings related to juvenile arthritis, ACL reconstruction, orthopaedic procedures, and perioperative care was used to identify articles with potential relevance. In addition, recent cases of ACL reconstruction in patients diagnosed with JIA will be identified from the health records of BC Children's Hospital and reviewed for trends in JIA management and surgical outcomes.

Preliminary Results

A total of 1688 references were identified by the initial search, with 1296 references remaining after the removal of duplicates. Title and abstract screening by a single reviewer yielded 351 articles for full-text review. Progress to date has identified 48 studies mentioning the perioperative medical management of JIA and/or the complication risk associated with unmanaged JIA in the perioperative window.

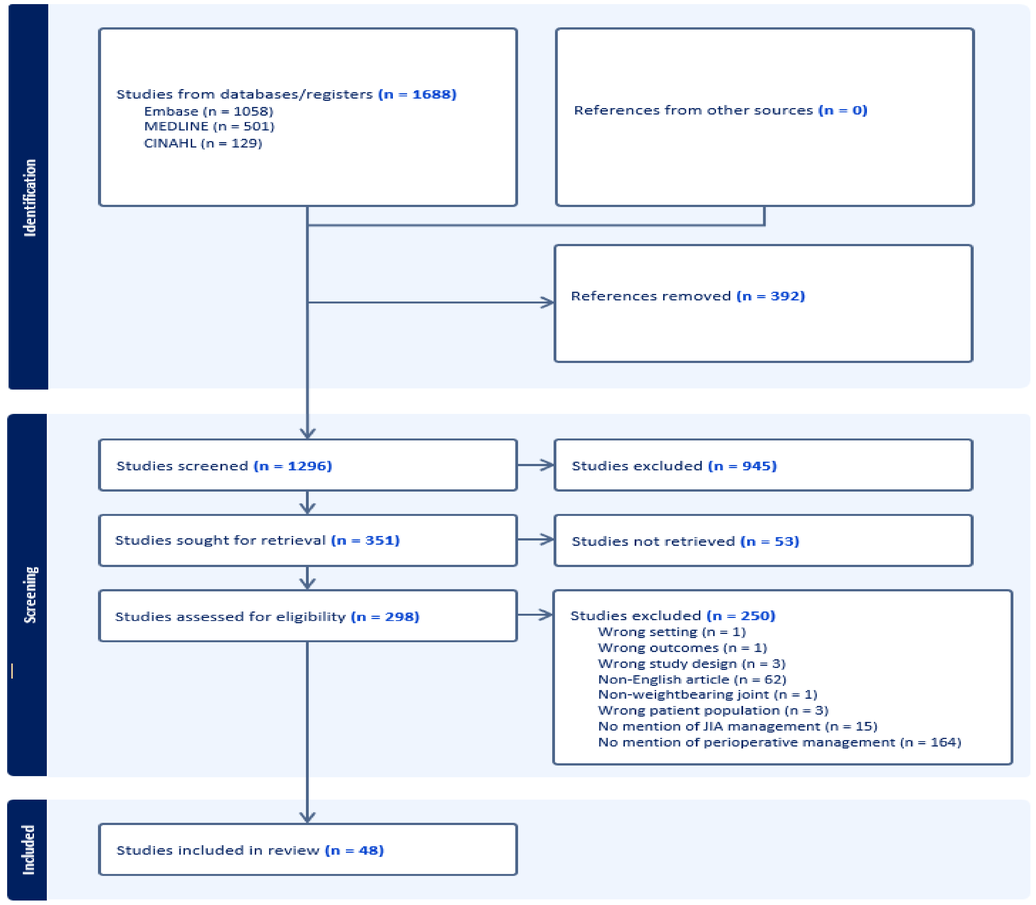


Figure 1: PRISMA Flowchart for Multi-Database Literature Review

From a structured search of MEDLINE, Embase and CINAHL, 48 studies mentioning perioperative medical management of JIA and/or JIA-related complication risk have been identified.

Implications

The findings of this study may serve to guide the management of DMARDs, corticosteroids, and other antirheumatic medications during the perioperative period of paediatric ACL reconstruction. Optimal medical management of JIA in the perioperative window could serve to improve surgical outcomes and reduce complication rates for paediatric ACL reconstruction and other orthopaedic procedures.

#8

Amrith Vincent

Medical Student, Royal College of Surgeons in Ireland

Supervisor: Linlea Armstrong

Path to Progress: Managing Leukemia Predisposition in Pediatric Patients

Path to Progress: Managing Leukemia Predisposition in Pediatric Patients

Amrith Vincent [1,2,5] , Audi Setiadi [2], Adrienne Elbert [1], Alison M.W Castle [1], Kim Seath [1], Kristal Louie [1], Katherine Blood [3,4], Caron Strahlendorf [2], Linlea Armstrong [1]

1.BC Women's Hospital 2. BC Children's Hospital 3. BCCA Hereditary Cancer Program 4. VIHA Medical Genetics, 5. Royal College of Surgeons In Ireland

Background

Leukemia is one of the most common childhood cancers, accounting for 29%. Most pediatric leukemia patients have sporadic somatic variants. Some are at risk due to germline variants. It is estimated that nearly 10% of pediatric cancer patients have a germline variant in a cancer predisposition gene. The leukemia variants affect key regulatory processes at the cellular level, such as DNA repair and genomic stability. The most common leukemia predisposition genes are somatically changed in other cancers (eg. TP53, RUNX1, IKZF1, and ETV6).

Objective

Our goal is to create a comprehensive care pathway that allows clinicians to identify and provide personalized care plans to patients and families with a pediatric leukemia predisposition syndrome.

Recognizing predisposition syndromes are important due to differences in clinical management, the need for genetic counseling and for hematopoietic stem cell donor selection.

Methods

A comprehensive literature search was performed using the PubMed database and internet search engines. Search terms included "leukemia," "pediatric," "predisposition," and "therapeutics." Exclusion criteria included adult only predisposition syndromes.

Conclusions

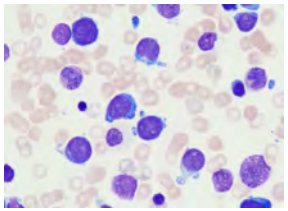
The care pathway will be a valuable resource for clinicians navigating the complex landscape of pediatric leukemia predisposition syndromes, providing the blueprint for the creation of personalized care plans throughout the patient's journey.

[1]

Care pathway

Recognition

- Clinical features: age, family history of conditions such as bone marrow failure, aplastic anemia etc.
- Physical exam findings could include features such as variations in growth, skeletal malformations and skin/mucosal/hair changes



Bone marrow aspirate showing acute megakaryoblastic leukemia associated with Down syndrome (50x)

Cancer Predisposition Syndrome Diagnostic Assessment

- Review of historic CBC's, Hemoglobin electrophoresis
- Bone marrow biopsy, with morphology, cytogenetic analysis, and molecular testing
- Trypsinogen, pancreatic isoamylase
- Erythrocyte ADA
- Liver transaminases
- Immunoglobulin levels
- Somatic and / or germline genetic testing
  - i. Metaphase cytogenetic analysis
  - ii. Chromosomal microarray
  - iii. Single nucleotide polymorphism array
  - iv. Fluorescent in situ hybridization (FISH)
  - v. Next-generation sequencing

Management of the Cancer Predisposition Syndrome

- Genetic counselling
- Specific management varies between specific syndromes
  - Treatment
  - Diagnostic Imaging
  - Surveillance
- Management for the family

Transition to Adult Care

- Address needs and knowledge gaps of patient and families through transition tools
- Early start on education of patient
- Communication between healthcare providers
- Multidisciplinary conferences

Acknowledgements

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#9

Thumri Waliwitiya

Medical Student, University of British Columbia

Supervisor: Tim Oberlander

Game Based Intervention for Improving Executive Function in Children with Congenital/Acquired Heart Disease

Game Based Intervention for Improving Executive Function in Children with Congenital/Acquired Heart Disease

Thumri Waliwitiya[1], Sarah M. Hutchison[1], Yaewon Kim[2], Buse Bedir[2], John Sheehan[2], Astrid De Souza[1], Kathryn R. Armstrong[1], Sarah J. Macoun[2], and Tim F. Oberlander[1]

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Introduction

Many children with congenital heart disease (CHD) are at risk of experiencing *executive function* (EF) difficulties. EF skills help one to control actions, thoughts, and emotions, as well as to plan, organize, and execute complex tasks. They are critical for academic and psychosocial success, self-care, and successful transition from the pediatric to adult medical system. Children with CHD may have problems with working memory, inhibition, and shifting in performance-based tasks.

Recently there has been interest in delivering online cognitive interventions. There is mounting evidence that computerized attention/EF training can be effective for developmentally and neurologically diverse populations if delivered appropriately.

Study Objectives

1. To work with stakeholders (CHD patients and team) to administer the EF intervention.

2. To determine the feasibility of using this intervention, the level of engagement, and satisfaction of CHD patients, their family, and their CHD team with the EF intervention.

3. To measure EF, and brain function in children with CHD. To measure quality of life (QoL) of children and their families pre- and post-implementation of the EF intervention.

"The video game is a treatment program that kids will stick with long enough to make it effective and doesn't require a clinical expert to deliver it"

- Dr. Sarah Macoun

"You go from this really simple switching where the game just tells them what to do, to having to monitor and figure it out themselves, which gets into more of the executive functioning and higher order aspects"

- Dr. Sarah Macoun

Methods

Recruitment and Pre-Training Assessment

- Letter of invitation sent to all eligible participants in the BCCH Heart Center
- Pre-training assessment at BCCH, Online training for parents on intervention delivery
- Outcomes measured by: Verbal intelligence, Nonverbal intelligence, Verbal and spatial working memory, Cognitive flexibility, Spatial working memory, Sustained attention,

Game Play

- Participants play Dino Island for 6 weeks
- 3 40-60 min sessions/week

Post-Training Assessment

- Outcome assessment
- Collection of feedback from participating families on their experience

Significance

- As most children with CHD survive into adulthood, there has been a shift to optimize long-term outcomes in these patients.
- Our study aims to improve EF in children with CHD using innovative game-based intervention which has shown to be effective in other clinical populations with poor EF skills.
- Improving EF and academic skills may be beneficial for long-term health outcomes and QoL in children with CHD.

T. Waliwitiya was supported during this work as it was generously funded by grants from the BC Children's Hospital Research Institute.

This work was conducted out of Vancouver, Canada on the traditional, ancestral, and unceded territories of the xʷməθkʷəy̓əm (Musqueam), Sḵwx̱wú7mesh (Squamish), and Sel̓íl̓witulh (Tsleil-Waututh) Nations.


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